CASE REPORT

Thrombosis of Portal Venous System after Laparoscopic Cholecystectomy in a Patient with Prothrombin Gene Mutation

Waheed Gul, MD, Khurram Abbass, MD, Arif M. Qazi,
Ronald J. Markert, PhD, Christopher J. Barde

ABSTRACT

Laparoscopic cholecystectomy is now the gold standard for the treatment of symptomatic cholelithiasis. Portal venous thrombosis after laparoscopic cholecystectomy is rare. We report a case of thrombosis of the portal venous system after laparoscopic cholecystectomy in a patient with a latent prothrombin gene mutation. An abdominal computed tomography and magnetic resonance angiogram of the abdomen revealed portal, superior mesenteric, and splenic vein thrombosis. Testing for coagulation disorders showed a heterozygous form of factor II (prothrombin) G20210A mutation. Because of its rarity, information regarding this complication is limited.

Key Words: Portal venous thrombosis, Laparoscopic cholecystectomy, Prothrombin gene mutation.

INTRODUCTION

Portal venous thrombosis (PVT) refers to the development of thrombosis within the extrahepatic portal venous system draining into the liver. Most cases of PVT have an identifiable cause related to a thrombophilic disorder or local factors. Local factors, such as infection, inflammation, portal venous system injury due to surgeries, or impaired portal vein flow, act as a thrombogenic stimulus.[1] Local factors explain why thrombosis suddenly develops in the portal venous system in a patient with an undiagnosed latent state of thrombophilia.[2] In patients with PVT, thrombophilic disorders are identified in approximately 60% and a local factor in 40%.[3] Here we share our experience with an unusual case of PVT after laparoscopic cholecystectomy (LC).

CASE REPORT

A 55-year-old Caucasian female underwent LC for symptomatic cholelithiasis. Her past surgical history revealed a right inguinal hernia repair, total abdominal hysterectomy, and bilateral salpingo-oophorectomy with an uncomplicated postoperative course. Approximately 2 months after LC, the patient started to experience diffuse intermittent abdominal pain. Two days before admission to our hospital, she had worsening of abdominal pain, bloating, and vomiting.

Physical examination revealed diffuse abdominal tenderness. The results of laboratory testing at the time of admission were as follows: leukocyte count 15 400/mm^3 (normal range, 3.8 to 10.8), amylase <30u/L (normal range, 0 to 110), lipase 18u/L (normal range, 0 to 60), prothrombin time 10.6 seconds (normal range, 8.5 to 11.9), and normal liver function tests. Plain abdominal radiograph revealed air fluid levels within the small bowel with a nonspecific bowel gas pattern. An abdominal computed tomography (CT) revealed portal and mesenteric vein thrombosis and multiple poorly defined, low-density infiltrative lesions in the liver. Magnetic resonance angiogram (MRA) of the abdomen showed portal and splenic vein thrombosis **(Figure 1)**. The patient underwent CT-guided biopsy of the liver. Histology showed benign hepatic tissue with no inflammation.

Dayton Veteran's Affairs Medical Center, Dayton, Ohio, USA (Dr. Abbass); Wright State University, Boonshoft School of Medicine, Dayton, Ohio, USA (all authors).

Address correspondence to: Khurram Abbass, MD, Dayton Veteran's Affairs Medical Center, Dayton, OH, USA and Wright State University, Boonshoft School of Medicine, Dayton, Ohio, USA. Telephone: (616) 334-5028, Fax: (937) 429-9050, E-mail: drkhuramabbass@gmail.com

DOI: 10.4293/108680812X13291597716663

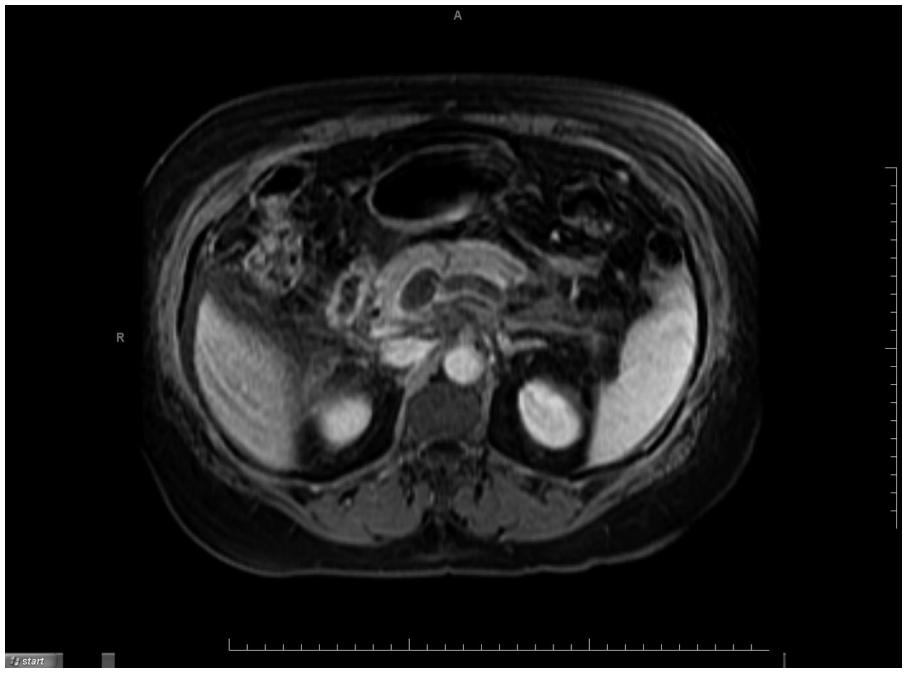

Figure 1. Magnetic resonance angiogram of the abdomen showed portal and splenic vein thrombosis.

Further testing showed a carcinoembryonic antigen level of 0.9ng/mL (normal range, <2.5), carbohydrate antigen 19U/mL to 96U/mL (normal range <40) and alpha-fetoprotein 1.6ng/mL (normal range <6.1). Testing for coagulation disorders showed levels of antithrombin III 108% (normal range, 79 to 139), protein C activity 93% (normal range, 64 to 168), protein S total 94.4% (normal range, 51.7 to 46.1), protein S free 96.3% (normal range, 50.4 to 75), homocysteine level 4.6umol/L (normal range, 4.9 to 14.8), and nondetected factor V Leiden.

The patient was found to have the heterozygous form of factor II (prothrombin) G20210A mutation. Lupus anticoagulant panel was negative. Flow cytometry was negative for paroxysmal nocturnal hemoglobinuria clone, and acute leukemia panel was also negative. Anticoagulation therapy with low-molecular-weight heparin was initiated and was later changed to oral anticoagulation with warfarin. The patient was discharged from the hospital on the seventh day. The result of abdominal Doppler ultrasound performed 6 weeks after discharge revealed complete dissolution of the thrombus.

DISCUSSION

PVT is a well-recognized complication after laparoscopic surgeries, such as splenectomy (55%), Nissan fundoplication, colectomy, appendectomy, and gastric bypass surgeries, but is rare after laparoscopic cholecystectomy.[4] It has been reported only twice in Medline.[5,6]The most likely cause of PVT after LC may be inflation of carbon dioxide with increased intraabdominal pressure, resulting in a change in coagulation status as well as splanchnic, hemodynamic, and portal venous blood flow.[5,6] It is now widely accepted that PVT occurs both from a primary thrombophilic disorder and a local factor that triggers the formation of pathologic thrombus in portal circulation. Valla et al[2] suggested, as a rule, this type of operation does not precipitate portal vein thrombosis unless there is an associated prothrombotic state or portal hypertension. In our patient, the thrombophilic disorder was factor II G20210A mutation, and most likely the local factor was capnoperitoneum-related alteration in coagulation and splanchnic hemodynamics.

The diagnosis of PVT can be accurately established by color Doppler ultrasonography, CT, or MRA. Color Doppler ultrasonography, the first line of investigation, is accurate, least expensive, and the least invasive diagnostic method. Treatment options include anticoagulation with heparin followed by warfarin, selective venography with infusion of thrombolytic agents, and surgical thrombectomy.[5,6] Anticoagulation therapy for at least 6 months should be considered and continued indefinitely if an underlying thrombophilic disorder is demonstrated. Our

patient with a factor II G20210A mutation required long-term anticoagulation.

CONCLUSION

Portal, mesenteric, and splenic vein thrombosis is an infrequent but serious complication of laparoscopic surgery. Here we present the first case of PVT caused by LC in a patient with prothrombin gene mutation G20210A. Interestingly, this patient had 2 open abdominal surgeries in the past with unremarkable postoperative courses. PVT should be suspected in patients who develop abdominal symptoms following laparoscopic surgery, even a few months following the procedure. The analysis of prothrombin gene mutation G20210A should be included in the workup of intraabdominal venous thrombosis, regardless of whether the patient has a personal or family history of venous thrombosis.

References:

1. van't Riet M, Burger JW, van Muiswinkel JM, Kazemier G, Schipperus MR, Bonjer HJ. Diagnosis and treatment of portal vein thrombosis following splenectomy. *Br J Surg*. 2000 Sep; 87(9):1229–1233.

2. Valla DC, Condat B. Portal vein thrombosis in adults: Pathophysiology, pathogenesis and management. *J Hepatol*. 2000 May;32(5):865–871.

3. Denninger MH, Chait Y, Casadevall N, et al. Cause of portal or hepatic venous thrombosis in adults: The role of multiple concurrent factors. *Hepatology*. 2000 Mar;31(3):587–591.

4. James AW, Rabl C, Westphalen AC, Fogarty PF, Posselt AM, Campos GM. Portomesenteric venous thrombosis after laparoscopic surgery: a systematic literature review. *Arch Surg*. 2009 Jun;144(6):520–526.

5. Preventza OA, Habib FA, Young SC, Penney D, Oppat W, Mittal VK. Portal vein thrombosis: an unusual complication of laparoscopic cholecystectomy. *JSLS*. 2005 Jan-Mar;9(1):87–90.

6. Balsarkar DJ, Sanjana MK. Unusual cause of pain in abdomen after laparoscopic cholecystectomy. *Indian J Gastroenterol*. 2008 Jan-Feb;27(1):37–38.